34. The method of claim 33, wherein said RTK-hyperfunction-induced disorder is one or more disorders selected from the group consisting of cancer, a disease attributable to cellular hyperproliferation and/or cellular invasion of tissue, a carcinoma, and a metastasis.

35. The method of claim 34, wherein said disorder is breast cancer, squamous cell carcinoma, glioblastoma, neuroblastoma or uterine cancer.

36. The method of claim 33, wherein said inhibitor is a kinase-inactive receptor.

37. The method of claim 33, wherein an overexpression and/or an altered activity of FGFR-4 is lowered and/or inhibited.

38. The method of claim 37, wherein the overexpression and/or the altered activity of FGFR-4 is triggered by a mutation of the FGFR-4.

39. The method of claim 38, wherein the mutation is one or several point mutations.

40. The method of claim 39, wherein the one or several point mutations leads to an exchange of a hydrophobic amino acid for a hydrophilic amino acid.

41. The method of claim 38, wherein the mutation occurs in the transmembrane domain of FGFR-4.

a' cont

42. The method of claim 41, wherein the mutation is one or several point mutations that lead to an exchange of a hydrophobic amino acid for a hydrophilic amino acid.

43. The method of claim 38, wherein the mutation occurs at amino acid position 388 in the FGFR-4 molecule.

44. The method of claim 43, wherein the mutation leads to an exchange of glycine for arginine.

45. The method of claim 38, wherein the mutation is a germ line mutation.

46. The method of claim 33, wherein the FGFR-4 is mutated and the inhibitor inhibits the mutated FGFR-4.

47. A mutated FGFR-4 which is overexpressed and/or has altered activity in a cell.

48. The mutated FGFR-4 of claim 47, in which a hydrophobic amino acid in the wild-type receptor has been exchanged for a hydrophilic amino acid in the mutated receptor.

49. The mutated FGFR-4 of claim 47, which comprises a point mutation in the transmembrane domain.

50. The mutated FGFR-4 of claim 49, in which the point mutation occurs at amino acid 388 and, optionally, results in replacement of glycine with arginine.

51. A DNA or RNA molecule encoding the mutated FGFR-4 of claim 47.

52. A DNA or RNA molecule encoding the mutated FGFR-4 of claim 48.

53. A DNA or RNA molecule encoding the mutated FGFR-4 of claim 49.

54. A DNA or RNA molecule encoding the mutated FGFR-4 of claim 50.

55. A method of diagnosing an RTK-hyperfunction-induced disorder or a genetic predisposition therefor in a mammal, which method comprises determining the presence of a mutated FGFR-4 protein or a nucleic acid encoding a mutated FGFR-4 protein in a sample of protein or nucleic acid, respectively, obtained from said mammal, wherein the presence of such a protein or nucleic acid is indicative of an RTK-hyperfunction-induced disorder or a genetic predisposition therefor.

56. The method of claim 55, wherein said RTK-hyperfunction-induced disorder is cancer.

57. The method of claim 55, which method comprises contacting the sample of nucleic acid with a labeled DNA or RNA molecule encoding a mutated FGFR-4 under hybridizing conditions and detecting the labeled DNA or RNA molecule after hybridization, wherein the detection of the labeled DNA or RNA is indicative of the presence of a nucleic acid molecule encoding a mutated FGFR-4 in the sample.

58. The method of claim 55, which method comprises contacting the sample of nucleic acid with a restriction enzyme whose recognition sequence is affected by the mutation in the mutated FGFR-4 and detecting the presence or absence of fragments or the presence of altered fragments of the nucleic acid after contact with the restriction enzyme, wherein the absence of fragments or the presence of altered fragments of the nucleic acid after contact with the restriction enzyme is indicative of the presence of a nucleic acid molecule encoding a mutated FGFR-4 in the sample.

59. The method of claim 58, wherein the mutation in the mutated FGFR-4 occurs at amino acid position 388.

60. A method of identifying an inhibitor of tyrosine kinase activity, which method comprises contacting a potential inhibitor with a mutated FGFR-4 and determining tyrosine kinase activity in the absence and presence of the potential inhibitor, wherein a decrease in tyrosine kinase activity in the presence of the potential inhibitor indicates that the potential inhibitor is an inhibitor of tyrosine kinase activity.

61. A method of treating cancer in a mammal, which method comprises administering to the mammal an effective amount of a kinase-inactive receptor or an inhibitor that inhibits FGFR-4, whereupon said cancer is treated.

62. An antibody that reacts specifically with the mutated FGFR-4 of claim 47.

63. An antibody that reacts specifically with the mutated FGFR-4 of claim 48.

64. An antibody that reacts specifically with the mutated FGFR-4 of claim 49.

65. An antibody that reacts specifically with the mutated FGFR-4 of claim 50.